CROSS-CANADA DISEASE REPORT

RAPPORT DES MALADIES DIAGNOSTIQUÉES AU CANADA

Ontario

Canine stress syndrome

Three dogs of sporting breeds susceptible to anesthetic-induced malignant hyperthermia (MH) developed exercise-induced canine stress syndrome (CSS; 1), which is analogous to porcine stress syndrome. Hyperthermia (>42°C), dyspnea, hyperlactatemia (>10 mmol/L) and apparent muscle cramping occurred to a degree disproportionate to the exercise intensity. Susceptibility was definitively diagnosed using the "caffeine contracture test" (2) and "lymphocyte calcium test". No other abnormalities were consistently detected prior to development of CSS.

Canine stress syndrome is a differential diagnosis for exercise intolerance in young dogs. **The anesthetic-induced form is often fatal, whereas the exercise-induced form is usually reversible.**

The syndromes are caused by a latent abnormality of intracellular calcium homeostasis which is triggered by anesthetics or muscle stimulation. Signs are attributable to uncontrolled activation of muscle metabolism and contraction.

An exercise challenge test was used in these dogs to screen for susceptibility to CSS. In response to ten minutes of moderate-intensity exercise, mild hemoconcentration and respiratory alkalosis occurred in addition to the clinical signs mentioned above. Although dantrolene prevents anesthetic-induced MH, it did not prevent exercise-induced CSS, which is likely initiated by a different subcellular pathway (3).

The "lymphocyte calcium test", a recently reported test for MH-susceptibility in swine and people, was found to be effective in dogs.

References

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Saskatchewan

Suspected oriental mustard seed (*Brassica juncea*) poisoning in cattle

In central Saskatchewan, we have observed acute mustard seed toxicosis similar to that described in southern Saskatchewan (1). During October of 1988, three of 60 cows in a Charolais herd developed bloody diarrhea, and died 24 h later. One cow that was submitted to the Western College of Veterinary Medicine had developed this bloody diarrhea two to three days after the ingestion of an unknown amount of commercially grown oriental mustard (*Brassica juncea*). The seeds had been swept off a combine and dumped in piles on the pasture where the animals had been grazing.

One animal was examined postmortem. The 12 year old cow was in good bodily condition but severely dehydrated, with marked soiling of hindquarters by frank blood and fecal material. Lesions were confined to the abdominal cavity where there was reddening of serosal surfaces and multifocal ecchymotic hemorrhages of the cecum and colon, and edema of the small intestine. Several mucosal necrosis and hemorrhage of the cecum and colon were characterized by frank blood and extreme mucosal edema; luminal blood clots contained fragmented mustard seed. The abomasal mucosa was mildly edematous, but the reticulum and rumen were normal. No infectious agents were isolated from this animal, and large amounts of oriental mustard seed (*Brassica juncea*) were seen in the rumen, abomasum, and large intestine.

Brassica juncea is part of the family Cruciferae, or the mustard group of plants, which often contain large amounts of volatile mustard oils in the glycosidic form, referred to as glucosinolates. It is the enzymatic degradation product of these glucosinolates, the volatile mustard oil, which is the toxic principle responsible for gastrointestinal poisoning (2). In this case, *B. juncea* contains the glucosinolate sinigrin, which, in the presence of moisture and the enzyme myrosinase, yields allyl isothiocyanate (an irritant and potentially toxic mustard oil), potassium hydrogen sulphate, and glucose.

The glucosinolates and myrosinase are located in different cells within the seed. For this reason, the plants are harmless until the cells of the seeds are moistened, crushed, and acted on by ruminal microflora. Glucosinolates and myrosinase may then combine to form the volatile mustard oil. Feeding experiments with mustard cake of known allyl isothiocyanate content have established a lethal dose in cattle of 0.001% of the animal's weight in oil (2).

It is possible that the difference in lesions observed between the two Saskatchewan cases may be explained by the amount of seed consumed. Large amounts of mustard seed may result in the rapid release of allyl isothiocyanate in the forestomachs, whereas lesser amounts may not release enough allyl isothiocyanate to cause much tissue damage until it is concentrated in the large intestine following the resorption of water.

Reported cases of poisoning in livestock from consumption of this plant are few, but nevertheless significant. These cases highlight the importance of dispersing mustard seed following harvest before cattle are let on to the pasture.

References

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Acute neonatal actinobacillosis in calves

A systemic infection was the cause of death of two young calves from two unrelated premises. One was a four-day-old Charolais, the other a five-day-old Aberdeen Angus. The lesions found in the Charolais calf were vegetative endocarditis, severe fibrinous polyarthritis, embolic suppurative nephritis, and embolic pneumonia; the Angus calf had embolic nephritis, hepatitis, pneumonia, adrenalitis, and encephalitis. Histopathological studies revealed bacteria in many of the lesions, often plugging small blood vessels, notably the renal glomerular and pulmonary capillaries.

In the Angus calf, *Actinobacillus lignieresii* was isolated in pure culture from the brain, liver, spleen, and heart, and with *Escherichia coli* from the kidney. In the Charolais calf, *A. lignieresii* was isolated in pure culture from the kidney, lung, and joint aspirate, and with non-hemolytic *Streptococcus* sp. from the heart.

The identification of the organisms was done by standard biochemical tests. For the isolate from the Charolais calf, only basic tests were done, whereas a thorough study was done on the organism isolated from the Aberdeen Angus. This bacterium produced acid from glucose, maltose, raffinose, sucrose, mannitol, xylose, and lactose (slow), but not from trehalose, salicin, rhamnose, adonitol, inositol, or sorbitol. It produced urease and H_2S, was ONPG positive, and negative for esculin hydrolysis, gelatinase, lysine decarboxylase, indole production, and citrate utilization. Both strains produced "sticky" colonies on blood agar, and none grew on MacConkey's agar.

From a comparative pathological standpoint, the lesions are very similar to lesions frequently encountered in newborn foals infected with *Actinobacillus equuli*.

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British Columbia

Plague in bushy-tailed woodrats

Two bushy-tailed woodrats (*Neotoma cinerea*) from the Lillooet area of B.C. were submitted frozen for examination in late January, 1989. Woodrat populations in that area were depleted from the previous winter. These animals were found dead in August and October of 1988 and collected from one of several study sites. The animals were in poor condition but had been eating. *Yersinia pseudotuberculosis* var. *pestis* was recovered from internal organs. **This is the first recovery of *Yersinia pseudotuberculosis* var. *pestis* in B.C. and only the second time it has been isolated in Canada.**

Bubonic plague was diagnosed in a trapper in Washington state in 1984; as a result, the B.C. Ministries of Environment and Health began a monitoring program to determine the possible presence of plague in animals in B.C. Blood samples collected by trappers were sent to the Plague Reference Laboratory in Colorado for serological testing. In 1987, 3 of 160 animals tested positive for exposure to this agent; in 1988, all 200 samples were negative.

Plague is a disease transmitted primarily by flea bites; however, infection can also occur following handling of infected tissue. Fleas on woodrats in B.C. tend not to bite humans. The disease responds well to treatment with appropriate antibiotics. Wild and domestic carnivores, except felids, appear quite resistant to disease caused by the plague bacillus. In enzootic areas, primarily in the southwestern U.S., several cases of bubonic plague have been described in domestic cats.

Physicians and veterinarians in B.C. have been made